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FILE 'HOME' ENTERED AT 12:35:44 ON 21 JAN 2003

=> file caplus, medline, biosis
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FULL ESTIMATED COST

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=> s ozone?

L1 83736 OZONE?

=> s 11 and 12

L3 16 L1 AND L2

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PROCESSING COMPLETED FOR L3

L4 14 DUP REM L3 (2 DUPLICATES REMOVED)

=> d 1-14 ab,bib

L4 ANSWER 1 OF 14 CAPLUS COPYRIGHT 2003 ACS

AB New methods of manuf., uses and applications for nutraceuticals, their compds., exts., enzymes, and special new properties, including, but not limited to, anthocyanidins, pro-anthocyanidins, dimers, polymers, cryst. proanthocyanadin, leucocyanin, leucodelphinin, flavonoids, and polyphenols, hereafter coined "ultra-nutraceuticals". These new methods, applications, properties and uses offer superior environmental, performance, medical and economic alternatives to chem., synthetic and other natural-based wet/dry regular or concd. compds. from herbs, vegetables, fruits and natural fibers. These ultra-nutraceuticals are desired in the manuf. of, but not limited to, pharmaceuticals for treatment and prevention of disease, medical: wound dressings, surgical gowns/drapes; film barriers: wet/dry soaps, cleaning solns., sprays and surface coatings; deodorants/antiperspirants; nonwovens, including

moist/dry sheets & towels, feminine hygiene, incontinence and diapers; cosmetics; personal care; animal bedding and pet litter; agricultural sprays; compns. of matter such as sponges, paper and molded pulp products, and meat/poultry/fruit trays. New application for toothbrush sanitizer in the small appliance category. New appliance consists of an elec., hand-held toothbrush sanitizer fueled by ozone, which can be used with or without toothpaste. Ozone elec. toothbrush removes the need for toothpaste as ozone kills 99.9% of all bacteria in mouth within two seconds. Ozone is a safe, natural antibacterial chem. as compared to Colgate's Total toothpaste which contains triclosan as an active ingredient for antimicrobial effectiveness. Sales of this elec. toothbrush could erode global toothpaste sales affecting the markets of leading toothpaste manufacturers Colgate and Procter & Gamble. 2002:889393 CAPLUS 137:375074 Ultra-nutriceuticals and toothbrush sanitizer (ozone) Joyce, Catherine U.S. Pat. Appl. Publ., 5 pp. CODEN: USXXCO Patent English FAN.CNT 1 PATENT NO. KIND DATE APPLICATION NO. DATE ---------------US 2002172707 A1 20021121 US 2001-848590 20010503 PRAI US 2000-201492P P 20000503 ANSWER 2 OF 14 CAPLUS COPYRIGHT 2003 ACS A method of increasing glutathione levels in mammalian cells comprising administering an oral bolus of encapsulated pharmaceutically stabilized glutathione in a rapidly dissolving formulation to a mammal on an empty stomach. Pharmaceutical formulations including glutathione are also disclosed. A combination pharmaceutical is provided to ameliorate the detrimental effects of acetaminophen, a drug which consumes glutathione in the liver during metab., and in excess doses causes liver damage due to oxidative damage. The compn. includes 500 mg L-glutathione, 250 mg cryst. ascorbic acid, and 350 mg acetaminophen. 2002:736715 CAPLUS 137:253031 Pharmaceutical preparations of glutathione and methods of administration Demopoulos, Harry B.; Seligman, Myron L. U.S. Pat. Appl. Publ., 26 pp., Cont.-in-part of U.S. 6,350,467. CODEN: USXXCO Patent English FAN.CNT 3 PATENT NO. KIND DATE APPLICATION NO. DATE ----------US 2002136763 A1 20020926 US 2002-83327 20020225 WO 9829101 A1 19980709 WO 1997-US23879 19971231 AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG B1 US 6350467 20020226 US 1999-331947 19990628 P PRAI US 1996-34101P 19961231

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WO 1997-US23879 W 19971231 US 1999-331947 A2 19990628

- L4 ANSWER 3 OF 14 CAPLUS COPYRIGHT 2003 ACS
- Exposure to complex mixts. of air pollutants produces inflammation in the AB upper and lower respiratory tract. Since the nasal cavity is a common portal of entry, respiratory and olfactory epithelia are vulnerable targets for toxicol. damage. This study evaluated, by light and electron microscopy and immunohistochem. expression of nuclear factor-kappa beta (NF-.kappa.B) and inducible NO synthase (iNOS), the olfactory and respiratory nasal mucosa, olfactory bulb, and cortical and subcortical structures from 32 healthy mongrel canine residents in southwest metropolitan Mexico City (SWMMC), a highly polluted urban region. Results were compared to those in 8 dogs from Tlaxcala, a less polluted, control city. In SWMMC dogs, expression of nuclear neuronal NF-.kappa.B and iNOS in cortical endothelial cells occurred at ages 2 and 4 wk; subsequent damage included alterations of the blood-brain barrier (BBB), degenerating cortical neurons, apoptotic glial white matter cells, deposition of apolipoprotein E (apoE)-pos. lipid droplets in smooth muscle cells and pericytes, non-neuritic plaques, and neurofibrillary tangles. Persistent pulmonary inflammation and deteriorating olfactory and respiratory barriers may play a role in the neuropathol. obsd. in brains of these highly exposed canines. Neurodegenerative disorders, e.g., Alzheimer's, may begin early in life with air pollutants playing a crucial role.
- AN 2002:465482 CAPLUS
- DN 137:205371
- TI Air pollution and brain damage
- AU Calderon-Garciduenas, Lilian; Azzarelli, Biagio; Acuna, Hilda; Garcia, Raquel; Gambling, Todd M.; Osnaya, Norma; Monroy, Sylvia; Del Rosario Tizapantzi, Maria; Carson, Johnny L.; Villarreal-Calderon, Anna; Rewcastle, Barry
- CS Curriculum in Toxicology, University of North Carolina at Chapel Hill, Chapel Hill, NC, 27599-7310, USA
- SO Toxicologic Pathology (2002), 30(3), 373-389 CODEN: TOPADD; ISSN: 0192-6233
- PB Taylor & Francis Inc.
- DT Journal
- LA English
- RE.CNT 116 THERE ARE 116 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT
- L4 ANSWER 4 OF 14 CAPLUS COPYRIGHT 2003 ACS
- AB The effect of the acute administration of .alpha.-tocopherol (VitE) on ozone-induced changes in brain catecholamine levels was studied. Results showed that a high dose of acute orogastric administered VitE prevents O3-induced changes in catecholamine levels in striatum. This could be mediated by an antioxidant action of VitE. The metabolites that are broken down by monoamine oxidase (MAO) were found to be markedly increased after O3 exposure. It suggests an increased MAO striatal activity, which in turn can destroy synaptic neuronal endings, as occurs in Parkinson's disease. Results provide evidence that O3 effects on brain catecholamine levels are mediated by free radicals and suggest that the possible role of VitE protection in such O3 effects is to break the chain reaction induced by free radicals, despite its interactions with other antioxidant substances. The effects of exposure to 03 on catecholamine striatal levels can be prevented by acute orogastric supplementation of VitE in rats.
- AN 2002:905533 CAPLUS
- TI Acute orogastric administration of alpha-tocopherol protects from ozone-induced changes in rat striatal catecholamine levels
- AU Gonzalez-Pina, R.; Alfaro-Rodriguez, A.; Castorena-Maldonado, A.; Morales Martinez, J. J.
- CS Laboratorio de Plasticidad Cerebral y Proliferacion Cellular, SSA,

Instituto de la Communicacion Humana-CNR, SSA, Mex.

- SO Proceedings of the Western Pharmacology Society (2002), 45, 59-61 CODEN: PWPSA8; ISSN: 0083-8969
- PB Western Pharmacology Society
- DT Journal
- English LA
- THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD RE.CNT 28 ALL CITATIONS AVAILABLE IN THE RE FORMAT
- ANSWER 5 OF 14 CAPLUS COPYRIGHT 2003 ACS L4
- AΒ Disclosed is a method for the administration of glutathione orally comprising the administration of a bolus of glutathione which is pharmaceutically stabilized and encapsulated. The glutathione is administered on an empty stomach. The preferred stabilizer is ascorbic acid. A preferred formulation of glutathione according to the present invention provides capsules for oral use contg. 500 mg reduced L-glutathione, 250 mg cryst. ascorbic acid, and .ltoreq. 0.9 mg magnesium stearate, in a gelatin capsule. The glutathione capsules were administered to HIV infected males with CD4+ cell counts of > 500, and clin. responses were seen in the PBM intracellular glutathione levels.
- AN 2000:874129 CAPLUS
- DN 134:32995
- TIPharmaceutical compositions of glutathione and methods of administration
- IN Demopoulos, Harry B.; Seligman, Myron L.
- Antioxidant Pharmaceuticals Corporation, USA PA
- U.S., 24 pp. SO
 - CODEN: USXXAM
- DΤ Patent
- LA English
- FAN.CNT 3

IAM.CNI J					
	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
ΡI	US 6159500	Α	20001212	US 1997-2100	19971231
	US 6204248	B1	20010320	US 1999-457642	19991209
	US 6423687	B1	20020723	US 2001-813247	20010319
PRAI	US 1996-34101P	P	19961231		
	US 1997-2100	B1	19971231		
	US 1999-331947	Α	19990628		
	US 1999-457642	A1	19991209		

RE.CNT 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4ANSWER 6 OF 14 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 1

ΑB A review with 46 refs. is given on an interdisciplinary evaluation of the etiol., pathogenesis, and exptl. treatments of retinitis pigmentosa (RP). It addresses a 10-yr controversy concerning the rate of progression of RP. One lab. has estd. remaining visual field to be lost at a rate of 4.6% per yr, whereas another lab. ests. loss at 16-18%. This large discrepancy and lack of consensus needs resoln., since they pose serious statistical and operational problems for evaluating exptl. treatment approaches to RP. The resoln. of the controversy offered in the paper is based on a model of RP in which the initial rate of loss of visual field (the induction phase) is much slower than the subsequent logarithmic 1st-order rate of loss. The rationale for this kinetic model is that loss of mitochondrial function, possibly due to RP-genetically-related radical processes, has to reach a crit. threshold value before the mitochondrial trigger of programmed cell death or apoptosis (i.e., the release of mitochondrial cytochrome c by the opening of the permeability transition pore, PTP) can be activated by an encounter with a 2nd, but kinetically const. causative stress factor - most likely a light-stress-related factor. In its essential (2-causal) aspects, this kinetic model for RP is identical to the kinetic theories that were proposed for the Gombertz human mortality plot. The described kinetic model for RP provides a soln. to the visual

field-loss controversy, since the 1st study was performed with a population contg. a greater no. of patients in the slow stage of RP than the 2nd. Another objective of the investigation was to identify possible mechanisms of how the numerous genetic mutations in the rods of RP patients could give rise to damaging free-radical reactions capable of triggering apoptosis through their adverse effects on mitochondrial function. Another reason for focusing on radical reactions in RP was to provide a rationale for the proposed use of an extensive array of antioxidants and nutritional supplements for stemming progression of RP. In particular, the investigation focuses on saving cone-dependent central vision, i.e. on saving cells not affected by the genetic problems of the rods, but cells which can become lethally damaged by a spill-over of radicals and related harmful chem. reactions occurring in the rods. 3rd objective deals with the development of a rationale for a new strategy for retarding RP. This involves the use of desmethyldeprenyl, a metabolite of the anti-Parkinson's drug, deprenyl. The rationale is, in part, based on an observation that desmethyldeprenyl exerts antiapoptotic activities in a variety of neurodegenerative disorders. The protective mechanism involves the overexpression of the anti-apoptotic bcl-2 gene, leading to higher concns. of bcl-2 proteins, which by binding to mitochondria inhibits the trigger mechanism of apoptosis - the opening of PTP and release of cytochrome C. At the same time, desmethyldeprenyl causes the underexpression of the pro-apoptotic bax gene, which via bax proteins facilitates the opening of the PTP. Both the anti-apoptotic and pro-apoptotic mechanisms appear to be mediated by the binding of desmethyldeprenyl to glyceraldehyde-3-phosphate dehydrogenase. Antiapoptotic effects can also be generated by the parent compd., deprenyl, when this is used daily in low concns. of 1-2 mg/100 kg body wt. Under these conditions, it appears that the anti-apoptotic metabolite, desmethyldeprenyl, predominates over the pro-apoptotic metabolites of deprenyl, L-methamphetamine and L-amphetamine. Methamphetamine is not formed if desmethyldeprenyl is administered directly and thus could give desmethyldeprenyl a pharmacokinetic advantage over deprenyl. However, desmethyldeprenyl is still an FDA-unapproved substance and the possibility that deprenyl may on its own have unique anti-apoptotic effects, because of its structural similarity to desmethyldeprenyl, cannot be excluded at the present time. The relevance of these observations to RP is suggested by the findings of a recent study, that the progression of RP in transgenic mice can be retarded by genetic overexpression of the bcl-2 gene. The possibility of achieving beneficial synergistic effects by simultaneously causing the underexpression of the bax gene was not investigated. Apoptotic mechanisms were also implicated in other ocular diseases: glaucoma, optic neuropathies, ischemia (e.g. retinal detachment), cataract, diabetic retinopathy, and macular degeneration. Consequently, studies of possible beneficial effects of deprenyl or desmethyldeprenyl are also warranted in these disorders. The paper concludes with a crit. evaluation of several exptl. therapeutic regimens in current use for RP: the Russian Encad program, hyperbaric oxygen, ozone, and traditional chinese medicine. This evaluation focuses on potential dangers of these treatments and on the use of inappropriate outcome measures.

- AN 2000:450291 CAPLUS
- DN 133:290519
- TI Etiology, pathogenesis, and experimental treatment of retinitis pigmentosa
- AU Baumgartner, W. A.
- CS Ianus Foundation, Malibu, CA, 90265, USA
- SO Medical Hypotheses (2000), 54(5), 814-824 CODEN: MEHYDY; ISSN: 0306-9877
- PB Churchill Livingstone
- DT Journal; General Review
- LA English
- RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT

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L4
     ANSWER 7 OF 14 CAPLUS COPYRIGHT 2003 ACS
     The title compds. I [R1 = H, alkyl, alkoxy, etc., and R4 = OH, or R1R4 =
AΒ
     CR5R6(CH2)pCH(OH)O, etc.; p=0-2; R5, R6=H, alkyl, etc.; R2=H,
     alkyl, alkoxy, etc.; R3 = H, alkyl, etc.] are prepd. The title compd. I
     [R1 = R2 = tert-butyl; R4 = OH; R3 = ethyl] showed ED30 of 2.6 mg/kg/day
     for 10 days in rats with adjuvant arthritis.
     1999:297410 CAPLUS
AN
DN
     130:311809
     Preparation and formulation of isothiazolidine dioxides as antirheumatic
TI
     agents
     Matsumoto, Saichi; Jyoyama, Hirokuni; Kakudo, Shinji; Hanasaki, Kohji;
IN
     Koizumi, Kenzo; Sakata, Tsuneaki; Suzuki, Ryuji
PA
     Shionogi & Co., Ltd., Japan
     PCT Int. Appl., 88 pp.
SO
     CODEN: PIXXD2
DT
     Patent
     Japanese
LA
FAN.CNT 1
     PATENT NO.
                    KIND DATE
                                         APPLICATION NO. DATE
                    A1 19990506 WO 1998-JP4774 19981022
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PΙ
     WO 9921844
        W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE,
            DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE,
            KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX,
            NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT,
            UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
        RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES,
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            CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
     CA 2306914
                      AA
                           19990506
                                        CA 1998-2306914 19981022
     AU 9896456
                      A1
                           19990517
                                         AU 1998-96456
                                                         19981022
    AU 741180
                      B2
                           20011122
    BR 9812982
                      Α
                           20000808
                                         BR 1998-12982
                                                          19981022
    EP 1026162
                      A1
                           20000809
                                         EP 1998-950333
                                                         19981022
           AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
            IE, FI
     TW 475928
                      В
                           20020211
                                         TW 1998-87117583 19981023
PRAI JP 1997-292517
                      Α
                           19971024
    WO 1998-JP4774
                      W
                           19981022
OS
    MARPAT 130:311809
RE.CNT 7
             THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD
             ALL CITATIONS AVAILABLE IN THE RE FORMAT
L4
    ANSWER 8 OF 14 CAPLUS COPYRIGHT 2003 ACS
AB
    The invention concerns the detn. of lipid oxidizability in biol. systems,
    e.g. in lipoproteins, by using diphenylhexatriene and its lipid-derivs. as
    markers for detecting the progress of oxidn. via the decreasing
     fluorescent signal. The method is used for cells, serum, and food samples
     for measuring the effects of oxidants or antioxidants.
AN
    2000:134517 CAPLUS
DN
    132:148749
ΤI
    Fluorometric determination of lipid oxidizability in biological systems
    using diphenylhexatriene
IN
    Hermetter, Albin; Hofer, Gerald; Lichtenberg, Dov
PΑ
    Austria
SO
    Austrian, 10 pp.
    CODEN: AUXXAK
DT
    Patent
LA
    German
FAN.CNT 1
    PATENT NO.
                   KIND DATE
                                        APPLICATION NO. DATE
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                                         -----
PΤ
                    B 19991025
   AT 405693
                                        AT 1994-1875
                                                        19941004
    AT 9401875
                    Α
                          19990215
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- L4 ANSWER 9 OF 14 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
- This book is a collection of works on the topic of reactive oxygen species AB in biological systems. The twenty-eight individually authored chapters are divided into eight parts: Part I- Introduction; Part II- General Biochemistry and Molecular Biology; Part III- Nitrogen Reactive Species; Part IV- Environmental Pro- and Antioxidants; Part V- Internal Pro- and Antioxidants; Part VI- Specific Tissues; Part VII- Pathological States and Aging; Part VIII- Conclusion. Each chapter contains an introduction, a comprehensive discussion of the topic, a conclusion or summary, and a list of references for further information. A few examples of chapter topics include the chemistry of reactive oxygen species, the regulation of mammalian gene expression by reactive oxygen species, and oxidative stress and Parkinson's disease. This text, which is indexed and illustrated with figures and tables, should be a valuable reference tool for researchers and scientists who are interested in the biological effects associated with reactive oxygen species.
- AN 1999:443445 BIOSIS
- DN PREV199900443445
- TI Reactive oxygen species in biological systems: An interdisciplinary approach.
- AU Gilbert, Daniel L. (1); Colton, Carol A.
- CS (1) National Institutes of Health, Bethesda, MD USA
- SO Gilbert, D. L.; Colton, C. A.. (1999) pp. xxv+707p. Reactive oxygen species in biological systems: An interdisciplinary approach. Publisher: Kluwer Academic Publishers PO Box 989, 3300 AZ Dordrecht, The Netherlands.
 - ISBN: 0-306-45756-3.
- DT Book
- LA English
- L4 ANSWER 10 OF 14 CAPLUS COPYRIGHT 2003 ACS
- Protection of mitochondria from oxidative damage due to natural or disease processes as well as by the effects of exogenous factors, e.g. incident sunlight, exposure via inhalation to oxidative environmental toxins, consumption of dietary oxidants, and oxidative-stress-inducing pharmaceuticals, exposure to radiation including radiation therapy, among others, is provided by a compn. comprising L-ergothioneine.

 L-Ergothioneine may be prepd. in a pharmaceutically-acceptable carrier to form an agent for topical application to the skin, and for orally or parenteral administration. Effective application and delivery of L-ergothioneine is enhanced by encapsulation in a liposome, a preferred embodiment. Diagnostic methods for detg. exposure and susceptibility to radiation, radical, and reactive oxygen species in mammals is also provided.
- AN 1998:603192 CAPLUS
- DN 129:198021
- TI Methods and compositions using L-ergothioneine for the protection of mitochondria and for diagnostic methods for determining exposure and susceptibility to radiation, radical, and reactive oxygen species
- IN Yarosh, Daniel B.
- PA Oxis International, Inc., USA
- SO PCT Int. Appl., 29 pp. CODEN: PIXXD2
- DT Patent
- LA English
- FAN.CNT 1

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PATENT NO. KIND DATE APPLICATION NO. DATE
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- PI WO 9836748 A1 19980827 WO 1998-US3352 19980220
 - W: AL, AU, BA, BB, BG, BR, CA, CN, CU, CZ, EE, GE, GW, HU, ID, IL, IS, JP, KP, KR, LC, LK, LR, LT, LV, MG, MK, MN, MX, NO, NZ, PL,
 - RO, SG, SI, SK, SL, TR, TT, UA, UZ, VN, YU, AM, AZ, BY, KG, KZ,

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MD, RU, TJ, TM
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             GA, GN, ML, MR, NE, SN, TD, TG
                                           US 1998-26198
     US 6103746
                            20000815
                                                             19980219
                      Α
                            19980909
                                           AU 1998-63325
                                                             19980220
     AU 9863325
                       A1
     AU 744523
                       B2
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     JP 2001513760
PRAI US 1997-38749P
                       P
                            19970220
     US 1998-26198
                       Α
                            19980219
                       W
                            19980220
     WO 1998-US3352
              THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD
RE.CNT 12
              ALL CITATIONS AVAILABLE IN THE RE FORMAT
     ANSWER 11 OF 14 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
L4
AN
     1996:472180 BIOSIS
DN
     PREV199699201736
     The hydroxyl radical: From chemistry to human disease.
ΤI
ΑU
     Lubec, Gert
     Univ. Vienna, Dep. Paediatr., Waehringer Guertel 18, A-1090 Vienna Austria
CS
so
     Journal of Investigative Medicine, (1996) Vol. 44, No. 6, pp. 324-346.
     ISSN: 1081-5589.
DT
     General Review
LA
     English
L4
     ANSWER 12 OF 14
                         MEDLINE
                                                         DUPLICATE 2
AB
     Inhaled by mice, ozone induced stronger free radical reaction in
     the organism and led to a series of changes similar to senility. In this
     way the senility mouse models were established to observe the changes of
     intestinal flora in senile mice. The senile mice were
     given the root of Astrogolus membraceus decoction orally. The results
     showed that the imbalance of intestinal flora in these mice was recovered.
AN
     96286804
                  MEDLINE
                PubMed ID: 8679084
DN
     96286804
ΤI
     Changes of intestinal flora in senile mouse models and the
     antagonistic activity of the root of Astragalus membraceus (Fisch) Bge.
ΑU
     Yan M; Song H; Xie N; Zhang L
CS
     Institute of Chinese Materia Medica, China Academy of Traditional Chinese
     Medicine, Beijing.
     CHUNG-KUO CHUNG YAO TSA CHIH CHINA JOURNAL OF CHINESE MATERIA MEDICA,
SO
     (1995 Oct) 20 (10) 624-6, inside backcover.
     Journal code: 8913656. ISSN: 1001-5302.
CY
     China
     Journal; Article; (JOURNAL ARTICLE)
DT
LA
     Chinese
     Priority Journals
FS
EM
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ED
     Entered STN: 19960828
     Last Updated on STN: 19960828
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L4
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     94090327
                 MEDLINE
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- L4 ANSWER 14 OF 14 MEDLINE
- There is a wealth of new knowledge regarding mechanisms of carcinogenesis AB and their interaction with senescence and environmental insults, particularly on the effects of UV irradiation on the skin. Innovations and advances in tissue culture techniques now permit in vitro studies of keratinocytes and other benign and malignant skin-derived cells. The ageing processes and cutaneous neoplasia, therefore, can now be studied at the cellular level. New insights regarding the interrelationship of ageing, environment and cutaneous neoplasia are close at hand. Depletion in the number of Langerhans cells and suppression of their function in ageing and UV-exposed skin may allow tumour cells to overcome the host's defence system. The potential increase in UV irradiation due to depletion of the ozone layer may increase the incidence of skin tumours. Carcinogenesis involves three distinct steps: initiation, promotion, and malignant conversion. The mechanism has been studied in mice, where it is suggested the c-ras oncogene may play an important role.
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